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An Address
ON
SURGICAL TREATMENT OF GASTRIC AND DUODENAL ULCER*

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IN the investigation of a case of suspected gastric or duodenal ulcer so many factors arise that the position of the surgeon has advanced materially from his earlier status of a mere craftsman carrying out the instructions of his medical colleagues. At the present time it is as important for him as for the physician to understand the meaning of symptoms, to be able to appreciate the importance of the various lines of investigation and to make his own decision as to the advantages in a particular case of medical or surgical treatment. The natural result of this has been the gradual development of team work in which the physician and the surgeon are taking up more and more similar attitudes in conjunction with the radiologist and bio-chemist. Moynihan stated the case very clearly when he said that he was a physician compelled to treat his cases surgically. In the investigation of a case the physician and the surgeon are indistinguishable and in their treatment there is a general agreement that surgery has no part in acute ulceration and that purely medical treatment has but poor chances of success in the chronic cases. So that when we come to the surgical treatment of gastric and duodenal ulcer you must understand that I refer to chronic ulcer only.

We must all agree that before it is possible to recognize the abnormal one must have a good knowledge of the normal, and the investigation of the normal stomach has been of great value and has led to some surprises.

Shape of the stomach.—It was not long before it was realized that the traditional stomach of the old text books did not exist in the living body, and soon a new shape, the J shape of stomach, was described and variations from this so-called normal J shape were looked upon as pathological. Recent work has shown the necessity of revising this opinion. We must first of all recognize that in the erect position the greater curvature is below the level of the iliac crests in 75 per cent. of healthy men and nearly 90 per cent. of healthy women, and the hepatic flexure below this level in over 50 per cent. of healthy women.

We know now that there are three types of stomach which can co-exist with perfect digestive function. The great majority of healthy people have what is called an *orthotonic* stomach, the proportion being about 80 per cent. of men and women. This stomach shows normal position, normal peristalsis and normal emptying rate, and also has normal secretion in the same proportion. The next type, the so-called *hypertonic* stomach, tends to lie obliquely from above downwards to the right, has well marked peristalsis and a rather rapid emptying rate. This is associated with a high acid content and is found in 17 per cent. of healthy men and only 7 per cent. of healthy women. Once more the secretion coincides with the type, i.e., about 15 per cent. with hyperchlorhydria. The third type, the *hypotonic*, has diminished peristalsis, a lower position (exaggerated J with a high pylorus) and a slower emptying rate. This is found in 4 per cent. of healthy men and 15 per cent. of healthy women and here again the secre-

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tion coincides with the type, about 5 per cent hypochlorhydria. Further investigation shows that the hypertonic stomach is found more commonly in broad chested vigorous people and the hypotonic in narrow chested people who take little exercise. It will be seen then that caution must be exercised in deciding whether or not abnormal gastric tone is pathological, and it must be considered in relation to the physical peculiarities and habits of the individual.

The motility of the stomach is estimated by the rate of emptying and this varies a little according as it is tested by the x-rays or by the Rehfuss tube, but it varies very much more according to the type of meal given. Thus beef and mutton leave the stomach more slowly than chicken and fish, bread leaves slowly and milk and cream may be held up for a long time, but a gruel meal will have left the stomach entirely in two hours. Then too there is the comparatively rapid emptying in the hypertonic or comparatively slow emptying in the hypotonic stomachs, a variation from the normal which may be common to the type of individual and must not in itself be considered pathological. Perhaps the most important factor in determining the emptying rate is the tone of the pyloric sphincter.

Secretory functions.—A great deal has been learnt of these functions since the routine use of the Rehfuss tube has been in operation. Rehfuss first described his method in 1914, but we had something else to think about at that time, and the method was not employed in England till the war was over. As you all know, the method consists in withdrawing the contents of the stomach at frequent intervals through a tube which shall be sufficiently small in diameter to be tolerated by the patient for any length of time.

The resting juice, that is the fluid present in the fasting stomach, is first withdrawn, then a simple test meal is given and a portion of the contents of the stomach is aspirated every fifteen minutes till gastric digestion is over. The specimens are received into numbered test tubes, filtered, and examined chemically. The main interest lies in the quantitative estimation of the acid content, whether free or combined, and in practice a normal acidity is recognized, a high normal is called hyperchlorhydria, a low normal hypochlorhydria, while if no acid secretion can be detected the terms achlorhydria and achylia are used; the former implying that though acid is secreted it is neutralized in some way or other, the latter implying that no acid is secreted. It was very soon noticed that acidity may vary considerably in healthy subjects and it was found that it corresponded very closely with the tone of the stomach and therefore with the type of individual; so that a healthy subject with a normal stomach will have a normal acidity, a healthy subject with a hypertonic stomach will have a high normal acidity and a healthy subject with a hypotonic stomach will have a low normal acidity. Neither hyperchlorhydria nor achylia are in themselves pathological, though of course they occur more commonly in diseased conditions. A regular sequence was recognized, the acidity of the resting juice falling when the meal was given and then gradually rising to its maximum in an hour and a half, then gradually falling to normal in two and a half hours. Then came the question as to the cause of this general reduction in acidity and it is now accepted that the main cause is a reflux of the alkaline duodenal contents; and so one sees that the behaviour of the pyloric sphincter is again a most important factor, for if there is spasm preventing duodenal reflux, the rising tide of acidity goes on unchecked and a high acid content results; if the pyloric sphincter is relaxed, duodenal reflux is encouraged and a low acid content results. An immediate difficulty arises to explain the high acid content in duodenal ulcer, for we know that in this disease, the stomach empties rapidly and almost continuously and one might imagine that with the pylorus practically always open duodenal reflux could occur and neutralization take place. This does not happen, however, because the tide sets so strongly from the stomach that regurgitation cannot take place and in addition the force of gravity is against it as the typical stomach in duodenal ulcer is of the cowhorn type obliquely placed in the abdomen from above downwards to the right. The opposite extreme is seen in cases of gastric cancer where frequently the stomach empties itself rapidly and yet there is achylia. The explanation of this is that in gastric cancer, acid secretion is reduced to a minimum or is even non-existent. Certain diseases are associated with constant

variations in gastric acidity and it is enough to mention the hyperchlorhydria of duodenal ulcer and gastric ulcer with pyloric spasm and the hypochlorhydria of many wasting diseases such as chronic pulmonary tuberculosis, and the achylia of pernicious anaemia and cancer of the stomach.

Gastric secretion is influenced also by the type of food ingested, thus beef and mutton and particularly the extractives of meat such as beef tea and soups, excite the secretion of HCl, and as these have very little protein to take up the acid, acidity is very high. Bread and milk cause less secretion and raw eggs cause hardly any, which accounts for the value of this form of food in patients suffering from acute gastric ulcer. Oils and fats actually decrease acid secretion and can therefore be given freely to gastric ulcer patients, but are not always well tolerated.

Duodenal reflux and pyloric sphincter control has always proved difficult to explain and elaborate theories have been advanced to account for it. It is probable after all, that sphincter control follows the ordinary law of peristaltic movement as laid down by Bayliss and Starling and that regurgitation follows the natural laws of gravity and the varying pressures in the pyloric antrum and the duodenum.

Now I wish to say a few words about *pain* as an evidence of faulty gastro-intestinal function. Pain is the most important symptom in all gastric disorders. It is the symptom which brings the patient to the doctor and the true interpretation of its character is one of the most valuable means of diagnosis. It is necessary to classify pain under several heads:— (1) The type of pain, whether aching, boring, colicky and so on, local or diffuse; (2) the relation of its onset to the intake of food; (3) causes of relief or exacerbation; (4) the occurrence of free intervals.

We are all well aware of the differences between the pain of gastric ulcer and cancer, between that of gastric ulcer in its local stages and after it has caused perigastritis and implicated other organs such as the liver and the pancreas; we try to differentiate, often with poor success, between the pain of duodenal or gastric ulcer and the referred pain of chronic appendicitis or cholecystitis. We know that pain is commonly induced by taking food in ulcer of the lesser curvature and relieved by taking food in duodenal ulcer, we know that relief can be obtained in most dyspepsias by vomiting or by taking bicarbonate of soda, and that relief or an increase of pain may result from certain postures. We know that in nearly all the dyspepsias, painful periods are followed by intermissions of perfect or almost perfect comfort and that generally the free intervals last longer than the painful periods. Unless we know, however, what is the cause of the pain we cannot place the proper interpretation on these variations. Two explanations were advanced and accepted for many years. First it was suggested that the pain was due to the actual contact of food with the ulcer, but this had to be abandoned when it was found that in duodenal ulcer, although the stomach contents passed into the ulcerated duodenum almost as soon as the food entered the stomach, pain rarely occurred until one or two hours had elapsed. Then it was suggested that the acidity of the gastric contents irritated the ulcer, but this had to be abandoned when Hurst failed, after administering large doses of a 5 per cent. solution of HCl, to cause pain in certain gastric ulcer patients. Another point is that the contents of the fasting stomach (resting juice) often have a high acidity and yet it is well known that pain rarely occurs in duodenal ulcer patients before breakfast. Gradually it began to become more and more evident that pain is due to disordered tonicity in the gastric musculature. Examination by the x-rays had shown that in ulcer of the lesser curvature there is commonly an associated contraction of the circular muscle fibres causing a characteristic notch in the greater curvature, and there is increased peristalsis and pyloric spasm. On the introduction of food, the stomach should dilate to accommodate the meal but this is prevented by the muscle contraction and probably also by some degree of infiltration of the muscle in the neighbourhood of the ulcer, and pain results. In duodenal ulcer on the other hand, the stomach, already in a condition of hypertonus, empties its contents at a rapid rate by hyperperistalsis, and it is only when the stomach is nearly empty and the already tired muscles have to make an extra effort to expel the remains of the meal that pain is experienced. The administration of

food at once relaxes the spasm and immediate relief is experienced. Relief is also obtained by the administration of bicarbonate of soda and this is due, not to neutralization of acidity, but to the effervescence and gas formation due to the action of the acid on the bicarbonate. This causes a temporary distension of the stomach and a consequent relief of muscular spasm. It must be a common experience that bicarbonate of soda gives more rapid relief than carbonate of bismuth and this is due to the fact that more gas is formed from the former. In carcinoma the pain is due to rigidity of the stomach wall and pyloric obstruction.

In certain cases of gastric and duodenal ulcer, pain is persistent and not influenced much by the intake of food. This symptom occurs in advanced cases and always means that the ulcer has penetrated deeply enough to cause perigastritis or even that the floor of the ulcer is formed by a neighbouring viscus. Perhaps the most characteristic of these pains is the boring pain felt in the back when a lesser curvature ulcer has penetrated into the pancreas. I had a patient under my care recently with a lesser curvature ulcer of huge size, the floor of which was formed by a shallow depression two inches by one inch in the liver and a typical egg cup depression in the pancreas. This patient had had for months a fixed pain in the right shoulder, and in the back between the scapulæ. When he came round from his anæsthetic after a partial gastrectomy, he at once exclaimed that his pains had gone, and he has had no return of them so far.

The subject of *referred pain* is an interesting one. I am thinking particularly of what is called appendix dyspepsia. It is typical of the chronic appendix that it gives rise to epigastric pain and that this pain may closely simulate gastric or duodenal ulcer. The pain is due to disordered gastric peristalsis and tends to simulate gastric ulcer in the hypotonic type and duodenal ulcer in the hypertonic type. The chief guide to differentiation lies in the tone of the abdominal muscles and it may be stated as an axiom that rigidity means a local cause while the absence of rigidity is in favour of the pain being referred from some other source.

And now a few words on the genesis of gastric and duodenal ulcers. It is some fifteen years ago since Dr. William Hunter, physician to Charing Cross Hospital in London, gave an address before McGill University on oral sepsis. He insisted that oral sepsis was the cause of many diseases and drove home his argument with a great wealth of pathological experience. His views are now universally accepted, especially as regards the relationship of oral sepsis and "indigestion." At the present day the first step taken by the medical man when consulted by a patient suffering from dyspepsia, is to investigate the nose, nasopharynx, and mouth in order to discover or exclude the presence of a septic focus. I have no doubt that the treatment of septic foci in these regions has been of enormous value in curing early cases of gastric and duodenal ulcer.

If sepsis in the upper alimentary tract is causative, it is possible that sepsis in the lower alimentary tract may have its part, so that infection reaches the stomach by the blood stream as the result of intestinal sepsis or chronic appendicitis. It seems probable that the same infective agent will cause gastric ulcer in the hypotonic type and duodenal ulcer in the hypertonic type.

With regard to the trend of modern thought on the treatment of gastric and duodenal ulcer, all are agreed that surgery has no place in the treatment of acute ulcer. Surgery confines itself to the chronic ulcer. At first, some twenty-five years or so ago, operation was done, excepting emergencies, for pyloric stenosis only; gastro-jejunostomy was the operation for choice, and the results were dramatically successful. As time went on gastro-jejunostomy began to be employed for other conditions where the pylorus was not constricted, in such conditions as duodenal ulcer and ulcer of the lesser curvature. I think it is not too much to say that the more the conditions under treatment differed from the original, the worse the results became, and the worst of all results followed the employment of gastro-jejunostomy in such conditions as gastroptosis and hypotonus. A reaction took place and it was then laid down that operation on the stomach must not be done unless an ulcer or its effects could be demonstrated. This was a considerable advance, but dissatisfaction was still experienced, for it was found that the proportion of cures in gastric ulcer after gastro-jejunostomy was much lower than in the duodenal ulcer and

indeed that the cures in gastric ulcer were mostly in cases when the ulcer was near the pylorus. Another difficulty arose, namely the possibility that a chronic gastric ulcer might take on malignant changes. These two considerations led to a demand for a direct attack on the ulcer if it were in the stomach, and excision of the ulcer was practised. It was soon found that this was followed by a high rate of recurrence, and it was then combined with gastro-jejunostomy but without any great improvement in results. Then some form of partial gastrectomy was introduced and at the present time partial gastrectomy is being employed more and more in chronic gastric ulcer. At the same time it was felt that gastric and duodenal ulcers might be secondary to some infective focus which, if left untreated, would lead to recurrence and it became the rule, not only to eliminate sepsis in the nose and mouth but also to examine, at the time of operation, the condition of other abdominal viscera, especially the appendix and the gall-bladder, and deal with any abnormality discovered.

With regard to duodenal ulcer, the position was less complicated, for gastro-jejunostomy led to a cure of over 80 per cent. of cases, and of course, there was no risk that the ulcer if left would take on malignant changes. But here again the proportion of failures, if not high, is considerable, and especially was it discovered that if bleeding had been a marked feature of the case, there was a tendency for it to recur after gastro-jejunostomy. Hohlbaum of Leipzig, writing in 1922, gave the proportion as 22 per cent., and Hurst in 1924 makes the proportion as high as 42 per cent.; and so there has been a movement in favour of a direct attack on duodenal ulcer also and this has taken the form of excision with gastro-duodenostomy or the much more extensive operation of gastro-duodenectomy. Finney's operation of pyloroplasty has never been much practised in England because we seem to find few cases in which it can be done, and we are inclined to think that in too great a proportion of cases adhesions form and the pylorus is drawn up and fixed under the liver, leading to pyloric obstruction for which gastro-jejunostomy has to be done after all. It is too early yet to get an idea of the late results in these radical operations and we cannot yet say whether the bugbear of gastro-jejunostomy, gastrojejunal ulcer, has been eliminated. Nor is it possible to give an approximate estimate of the immediate mortality, but there is no doubt that it is considerably higher than the mortality of gastro-jejunostomy which is less than 2 per cent. However, with experience, and particularly with the adoption of splanchnic anæsthesia as practised by Gordon Taylor, Professors Cade and Apperly, it is reasonable to hope that the immediate mortality will be considerably lessened.

It may interest you to hear the views current in England on the treatment of two complications of gastric and duodenal ulcer, namely, hæmorrhage and perforation.

It is usual to describe hæmorrhage under two heads, namely, acute hæmorrhage and severe repeated hæmorrhage, some of which have nothing to do with gastric or duodenal ulcer. I refer more particularly to cirrhosis of the liver, splenic anæmia and the condition first described by Sir William Hale-White to which he gave the name of "gastrotaxis."

It is obvious that for conditions such as these, operations on the stomach have no place. Indeed severe hæmorrhage so rarely occurs in chronic ulcer, compared with its frequency in other conditions, that it must not be looked upon as pathognomonic of ulcer. Then there is a large class of hæmorrhages occurring from acute ulcers. These cases often have a short history, indeed the hæmorrhage may be the first sign, and differential diagnosis may be very obscure. Neilson from Faber's clinic, 1897-1909, reports ten deaths in 528 cases, only one or two of which had definite symptoms of ulcer before the hæmorrhage. One is not tempted to operate on these cases as the lesion may be very difficult to find and even more difficult to treat. It is important, therefore, to consider the probable fate of these cases if treated medically. At a combined meeting of the Medical and Surgical Sections of the Royal Society of Medicine in London last year, a mass of evidence was produced to prove that under medical treatment the mortality is less than 4 per cent., while the mortality after operation was about 36 per cent. Some of these statistics were gathered before transfusion of whole blood was introduced, but even in later cases the mortality is heavy, so that there was a

very general agreement that medical treatment is indicated for hæmorrhage from acute ulcers. A very much more difficult problem presents itself in cases of severe repeated hæmorrhage where the diagnosis of a chronic ulcer is established. One has to realize that these cases are poor surgical risks already, that if a further hæmorrhage takes place surgical intervention may be the *coup de grâce*, and yet that to control hæmorrhage from a chronic ulcer which may have become widely adherent, a formidable operation may be necessary, as a single gastro-jejunostomy is useless in such cases. Here is a case for the very close co-operation of the physician and surgeon who must decide whether the chances are in favour of recovery or not. The first step is always to have the blood grouped and a suitable donor of blood arranged for. At the same time the hæmoglobin content is estimated. If it is below 30 per cent. transfusion is done at once. If it is agreed that success cannot be anticipated without operation, the question arises what operation should be done, and when it should be done. As regards the type of operation, it must be some form of partial gastrectomy designed to excise the bleeding area. Polya resection is probably the best, in a very favourable case a sleeve resection, and very rarely a local excision of the ulcer combined with a gastro-jejunostomy. One does, in fact, whatever operation will control the bleeding according to the power of resistance of the patient. Then arises the question of the best time to perform the operation. Let us agree that the best course is to put a stop to the hæmorrhage by medical means and do the radical cure three months later. In the particular cases under review that is impossible. It is agreed that operation is essential to save the patient's life. When should it be done? Should it be done while bleeding is actually occurring, or should one wait for a more favourable period. Gordon Taylor reported twenty-four cases operated on during the hæmorrhage with a mortality of two, i.e., 8 per cent. In sixteen cases he did a gastro-duodenal resection. This is a fine record but it is doubtful whether it is within reach of the average surgeon. Sherren is in favour of operating after the hæmorrhage has ceased, but says one must not wait more than forty-eight hours for fear of a recurrence. The question of transfusing or not is settled by the hæmoglobin content which must not be lower than 30 per cent. if success by operation is to be attained.

The treatment of perforation is becoming standardized. It is agreed on all hands that whatever is done the main factor for success is early recognition and early operation. Cases operated on within twelve hours should not show a higher mortality than 8 per cent., but the mortality advances with giant strides as delay increases. Discussion ranges round two considerations: (1) Whether to do a gastro-jejunostomy at the time of the closure of the perforation, and (2) whether to drain. Opinion in England agrees that perforation occurs generally in the chronic ulcer and that in something like 20 per cent. a subsequent operation had to be done for persistence of symptoms of ulcer. Therefore there seems to be a *prima facie* case for some more radical operation than mere closure of the perforation.

It must be remembered, however, that some of these perforations are in regions of the stomach where you would not choose gastro-jejunostomy as the curative operation, and therefore it is unreasonable to do it just because the ulcer is perforated. Again, though it is probable that when gastro-jejunostomy is done, it is done in the most favourable cases, yet it seems to be attended with a slightly higher mortality than simple suture. Murphy's dictum in abdominal emergencies "Quick in and quicker out" seems to me to be particularly applicable to these cases and I recommend simple suture at the time and a curative operation later on.

With regard to *drainage*, opinion is setting more and more in the direction of limiting its use to late cases. We have long since given up flushing the abdomen and local drainage is no longer used. If a drain is used it consists of a single drain passed to the floor of the pelvis. I am afraid I am old fashioned in this respect. I consider it essential to sit my patients up to limit the risk of sub-diaphragmatic abscess and it seems reasonable to provide some outlet for the fluid which must accumulate in the pelvis. So I drain the pelvis with a single tube for at most forty-eight hours and I believe it reduces the incidence of pelvic abscess.